Mouse Models for Cancer Research

The Jackson Laboratory offers 427 different strains for cancer research. A complete listing of these strains by research application is located on pages 2-10. More detailed information, including phenotypes and references, on Featured Mouse Models begins on page 11. Visit our JAX® Mice Database at www.jax.org/jaxmice/pricelist for more information, including price and availability. Search by research application or enter the stock number of the strain of interest and view the Strain Datasheet.

Newly Available Models

The supply of mice from strains that have recently become available for distribution is limited. Colony sizes are ultimately sized based on the broad needs of the research community. Please refer to the JAX® Mice Database for current availability and price information. If your experiments require numbers of mice that exceed our current supply, we will work with you to meet your needs (please contact our Customer Service Department or email JAX Breeding Services at jaxservices@jax.org).

New Strains Under Development Not Yet Available

Several of our featured strains are under development and will become available for distribution in the coming months. As an international repository and distribution center, The Jackson Laboratory makes available for distribution approximately 150 new mouse models each year. If a strain is not yet available for distribution, the JAX® Mice Database will refer you to a register interest form. Registering your interest will help us predict demand and set colony size. It will also ensure that you receive updates on the availability status with a formal notice about three weeks prior to distribution. Upon receiving advance notice, you will have the opportunity to place an advance order (*i.e.*, placement of an order prior to the strain being publicized as available). Advance orders are filled on a "first come - first served" basis in the original order that interest was registered.

JAX® Mice Website
www.jax.org/jaxmice

- Register for email notifications and updates on new models
- Search for JAX® Mice (datasheets, genotyping protocols, current pricing and availability)
- Explore newly available strains
- Register interest in new strains under development

Featured Models

MOUSE MODELS FOR CANCER RESEARCH

COMPLETE LISTING OF CANCER RESEARCH MODELS BY RESEARCH APPLICATION

The following list of JAX® Mice is designed to assist investigators in the selection of appropriate mouse models for cancer research. Investigators are strongly encouraged to research the specifics of any recommended mouse model prior to use to ensure that the recommended model is suitable.

Research application information is compiled using a number of sources, which are directly accessible from the JAX® Mice Web site at www.jax.org/jaxmice under the menu heading "Search for Mouse Information". These on-line sources include the Mouse Genome Database and Dr. Michael Festing's Inbred Strains of Mice and Rats. In addition, this list was prepared using McKusick's Online Mendelian Inheritance in Man and a review of the scientific literature.

♦ Chronic Myelogenous Leukemia

JAX® GEMM® Strains

Bcr
003803 STOCK *Bcr*^{tm1Hkp}

♦ Defects in Cell Adhesion Molecules

JAX® GEMM® Strains

Cdh3
003180 B6.129S-*Cdh3*^{tm1Hyn}

Ncam
002405 B6.129P2-*Ncam*^{tm1Cgn}

♦ Genes Regulating Growth and Proliferation

JAX® GEMM® Strains

Akp2
002484 129-*Akp2*^{tm1Sor}
002317 B6;129S-*Akp2*^{tm1Sor}
002741 B6.129S7-*Akp2*^{tm1Sor}

Asgr2
002387 129-*Asgr2*^{tm1Her}
002361 B6;129S-*Asgr2*^{tm1Her}

Bax
002994 B6.129X1-*Bax*^{tm1Sjk}

Bmp4
002612 B6.129S2-*Bmp4*^{tm1Blh}

Ccnd1
002537 B6;129S-*Ccnd1*^{tm1Wbg}
002935 FVB.129S2(B6)-*Ccnd1*^{tm1Wbg}

Cd38
003727 B6.129P2-*Cd38*^{tm1Lnd}

Cdc37
003690 STOCK TgN(MMTV-Cdc37)1Stp
003689 STOCK TgN(Pbsn-Cdc37)1Stp

Important Note

This list is not intended to be all-inclusive. Rapidly advancing biomedical research continually uncovers new applications for many strains, genes and gene mutations. For the most updated application information on JAX® Mice, please use the JAX® Mice searchable database at www.jax.org/jaxmice/pricelist.

JAX® GEMM® Strains

Genetically Engineered and Mutant Mice
These strains include transgenics and mice with spontaneous, chemically induced, or targeted mutations (i.e., "knockouts"). See the inside of the front cover for a complete description of JAX® GEMM® Strains.

JAX® Mice Database

JAX® Mice datasheets are available on the Web for all strains in this categorical listing from the JAX® Mice Database (www.jax.org/jaxmice/pricelist).

Genes Regulating Growth and Proliferation cont.

JAX® GEMM® Strains

Cdkn1a
003263 B6;129-*Cdkn1a*^{tm1Tyj}

Cdkn1b
003122 129/Sv-*Cdkn1b*^{tm1Mlf}
002781 B6.129-*Cdkn1b*^{tm1Mlf}

Cdkn1c
003336 B6.129S7-*Cdkn1c*^{tm1Sje}

Csk
003201 129/Sv-*Csk*^{tm1Sor}

Cycs
004264 B6;129-*Cycs*^{tm1Wlm}

Epas1
003266 B6;129-*Epas1*^{tm1Rus}

Fgf2
003256 STOCK *Fgf2*^{tm1Doe}

Fgf7
004161 B6;129-*Fgf7*^{tm1Efu}

Grpr
003126 B6.129X1-*Grpr*^{tm1Jfb}

HBV
002226 C57BL/6J-TgN(Alb1HBV)44Bri

IRS1
003268 FVB-TgN(IRS1)1Mhep

Igf1
002500 C57BL/6J-TgN(WapIgf1)39Dlr
003078 FVB-TgN(WapIgf1)39Dlr
003258 STOCK *Igf1*^{tm1Ts}
003259 STOCK *Igf1*^{tm2Ts}

Inhbb
002323 129S4/SvJae-*Inhbb*^{tm1Jae}
002442 B6.129S4-*Inhbb*^{tm1Jae}
002368 C.129S4(B6)-*Inhbb*^{tm1Jae}

Genes Regulating Growth and Proliferation cont.

JAX® GEMM® Strains

Inpp5d
003534 STOCK *Inpp5d*^{tm1Dmt}

Itga5
002274 B6.129S-*Itga5*^{tm1Hyn}

Kcna1
003532 C3HeB.129S7(B6)-*Kcna1*^{tm1Tem}

lacZ
002856 FVB/N-TgN(TIE2LacZ)182Sato

Map2k4
003666 B6;129-*Map2k4*^{tm1Liz}

Mdm2
002968 B6;129S-*Mdm2*^{tm1Bay}

NFKBIA
003330 B6;D2-TgN(LCK-NFKBIA)5Dwb

Ngfb
003312 B6.129S7-*Ngfb*^{tm1Agt}

Ntf3
002275 B6.129S4-*Ntf3*^{tm1Jae}
003541 B6.129S4-*Ntf3*^{tm2Jae}
002276 STOCK *Ntf3*^{tm1Jae}

Oxt
002713 B6;129S-*Oxt*^{tm1Wsy}

Pemt
003187 B6;129P-*Pemt*^{tm1J}

Plp
003255 B6;129-*Plp*^{tm1Kan}

Rac2
004197 B6.129S6-*Rac2*^{tm1Mddw}

Shh
003318 STOCK *Shh*^{tm1Amc}

Smst
003117 129S-*Smst*^{tm1Ute}

Src
002278 129-*Src*^{tm1Sor}
002381 B6;129S-*Src*^{tm1Sor}
002277 B6.129S7-*Src*^{tm1Sor}

Stat5a
002833 B6;129S-*Stat5a*^{tm1Mam}
003502 FVB.129S6-*Stat5a*^{tm1Mam}

Tcfap2a
002794 C.129S4-*Tcfap2a*^{tm1Jae}

TGFB1
002375 B6;D2-TgN(MMTVTGFB1)46Hlm
002933 FVB/NJ-TgN(MMTVTGFB1)46Hlm

Terc
004132 B6.Cg-*Terc*^{tm1Rdp}

♦ Growth Factors/Receptors/Cytokines

JAX® GEMM® Strains

Blmh
003509 B6.129-*Blmh*^{tm1Geh}

Bmp4
002612 B6.129S2-*Bmp4*^{tm1Blh}

Cd152
002980 C57BL/6-TgN(Cd152Ig)

Cmkar2
002724 C.129S2(B6)-*Cmkar2*^{tm1Mwm}

Cmkbr5
002782 B6;129P-*Cmkbr5*^{tm1Kuz}

Csf1^{op}
000231 B6C3Fe-*a/a-Csf1*^{op}

Csf3
002398 B6;129P-*Csf3*^{tm1Ard}

Egfr
002857 STOCK *Egfr*^{tm1Mag}

Egfr^{wa2}
000553 B6EiC3Sn-*a/A-Egfr*^{wa2} *Wnt3a*^{vt}
000317 STOCK *a/a Egfr*^{wa2}/+

Grpr
003126 B6.129X1-*Grpr*^{tm1Jfb}

Gzmb
002248 B6.129S2-*Gzmb*^{tm1Ley}
002247 B6;129S-*Gzmb*^{tm1Ley}

HBV
002226 C57BL/6J-TgN(Alb1HBV)44Bri

Ifng
002287 B6.129S7-*Ifng*^{tm1Ts}
002286 C.129S7(B6)-*Ifng*^{tm1Ts}
002294 D1.129S7(B6)-*Ifng*^{tm1Ts}

Ifngr
002702 129-*Ifngr*^{tm1Agt}
003288 B6.129S7-*Ifngr*^{tm1Agt}

Igf1
002500 C57BL/6J-TgN(WapIgf1)39Dlr
003078 FVB-TgN(WapIgf1)39Dlr
003258 STOCK *Igf1*^{tm1Ts}
003259 STOCK *Igf1*^{tm2Ts}

IGFBP3
002499 C57BL/6J-TgN(WapIGFBP3)67Dlr

Il1r1
003018 B6;129S-*Il1r1*^{tm1Roml}
003244 B6;129S-*Tnfrsf1a*^{tm1Imx} *Il1r1*^{tm1Imx}
003245 B6.129S7-*Il1r1*^{tm1Imx}

Il1rap
003284 B6;129S-*Il1rap*^{tm1Roml}

Growth Factors/Receptors/Cytokines cont.

JAX® GEMM® Strains

Il2
- 002252 B6.129P2-*Il2*^{tm1Hor}
- 002229 C.129P2(B6)-*Il2*^{tm1Hor}
- 002228 C3.129P2(B6)-*Il2*^{tm1Hor}
- 003862 NOD.B6-*D3Mit167-D3Mit94* (*Idd3, Il2*)
- 003852 NOD.B6-*Il2*

Il2ra
- 002952 B6.129-*Il2ra*^{tm1Dw}
- 002462 B6;129S-*Il2ra*^{tm1Dw}

Il2rb
- 002816 B6.129-*Il2rb*^{tm1Mak}

Il2rg
- 003174 B6.129-*Il2rg*^{tm1Wjl}
- 003169 C.129-*Il2rg*^{tm1Wjl}
- 002479 STOCK *Il2rg*^{tm1Cgn}

Il4
- 002253 B6.129P2-*Il4*^{tm1Cgn}
- 002496 BALB/c-*Il4*^{tm2Nnt}
- 003480 C.129S2(B6)-*Il4*^{tm1Gru}
- 002518 C57BL/6-*Il4*^{tm1Nnt}
- 002230 C57BL/6J-TgN(LckIl4)1315Dbl
- 002574 NOD.129P2(B6)-*Il4*^{tm1Cgn}/Dvs

Il4ra
- 003514 BALB/c-*Il4ra* ^{tm1Sz}

Il5
- 003175 C57BL/6-*Il5*^{tm1Kopf}

Il6
- 002254 B6;129S-*Il6*^{tm1Kopf}
- 002650 B6.129S6-*Il6*^{tm1Kopf}

Il7r
- 002296 B6;129S-*Il7r*^{tm1Imx}
- 002295 B6.129S7-*Il7r*^{tm1Imx}

Il10
- 002251 B6.129P2-*Il10*^{tm1Cgn}
- 002250 B10.129P2(B6)-*Il10*^{tm1Cgn}
- 003968 C3Bir.129P2(B6)-*Il10*^{tm1Cgn}
- 003089 NOD.129P2-*Il10*^{tm1Cgn}/Dvs

Il12a
- 002692 B6.129S1-*Il12a*^{tm1Jm}
- 002691 C.129S1(B6)-*Il12a*^{tm1Jm}

Il12b
- 002693 B6.129-*Il12b*^{tm1Jm}
- 002694 C.129-*Il12b*^{tm1Jm}

Il12rb1
- 002984 B6.129S1-*Il12rb1*^{tm1Jm}
- 003017 C.129S1-*Il12rb1*^{tm1Jm}

Il12rb2
- 003248 B6.129S1-*Il12rb2*^{tm1Jm}
- 003642 CBy.129S1-*Il12rb2*^{tm1Jm}

Growth Factors/Receptors/Cytokines cont.

JAX® GEMM® Strains

Kdr
- 002938 B6.129-*Kdr*^{tm1Jrt}

Kitl^{Sl} and alleles
- 000124 B6.Cg-*Ca Kitl*^{Sl}
- 000291 C3FeLe.Cg-*a/a-Ca*^{J} *Kitl*^{Sl} *Hm*
- 000693 WC/ReJ-*Kitl*^{Sl}/+
- 100401 WCB6F1/J *Kitl*^{Sl}/*Kitl*$^{Sl-d}$
- 001380 C3Sn.Cg-*Kitl*$^{Sl-con}$
- 000160 B6.D2-*Kitl*$^{Sl-d}$/+
- 000161 WB.D2-*Kitl*$^{Sl-d}$/+
- 100401 WCB6F1/J *Kitl*^{Sl}/*Kitl*$^{Sl-d}$
- 000090 129S1/Sv-*p*$^{+}$ *Tyr*$^{+}$ *Kitl*$^{Sl-J}$/+
- 000979 STOCK *Kitl*$^{Sl-16J}$
- 003252 C57BL/6J-*Kitl*$^{Sl-20J}$

lacZ
- 002856 FVB/N-TgN(TIE2LacZ)182Sato

Lifr
- 002402 B6;129S-*Lifr*^{tm1Imx}

Lta
- 002257 B6;129S-*Lta*^{tm1Dch}
- 002258 B6.129S2-*Lta*^{tm1Dch}

Map2k4
- 003666 B6;129-*Map2k4*^{tm1Liz}

Ncam
- 002405 B6.129P2-*Ncam*^{tm1Cgn}

Ngfb
- 003312 B6.129S7-*Ngfb*^{tm1Agt}

Ntf3
- 002275 B6.129S4-*Ntf3*^{tm1Jae}
- 003541 B6.129S4-*Ntf3*^{tm2Jae}
- 002276 STOCK *Ntf3*^{tm1Jae}

Ntf5
- 002497 129S4/SvJae-*Ntf5*^{tm1Jae}

Ntrk1
- 002480 B6;129S-*Ntrk1*^{tm1Bbd}

Ntrk2
- 002544 B6;129S-*Ntrk2*^{tm1Bbd}
- 003098 B6.129S2-*Ntrk2*^{tm1Bbd}

Ntrk3
- 002481 B6;129S-*Ntrk3*^{tm1Bbd}

Ph
- 000118 C57BL/6J-*Ph*

Prlr
- 003141 129X1/SvJ-*Prlr*^{tm1Cnp}
- 003142 B6.129P2-*Prlr*^{tm1Cnp}

Scya3
- 002687 B6.129P2-*Scya3*^{tm1Unc}

Growth Factors/Receptors/Cytokines cont.

JAX® GEMM® Strains

Shh
003318 STOCK *Shh*^{tm1Amc}

Tgfa
002219 B6.129P2-*Tgfa*^{tm1Ard}
002678 FVB/N-TgN(WapTgfa)215Bri

Tgfa^{wa1}
000004 ABP/Le
002863 B6.Cg-*Tgfa*^{wa1}

Tgfb1
002220 B6.129S2-*Tgfb1*^{tm1Doe}
002098 STOCK *Tgfb1*^{tm1Doe}

Tgfb2
003102 STOCK *Tgfb2*^{tm1Doe}

Tgfb3
002619 B6.129-*Tgfb3*^{tm1Doe}

Tnf
003008 B6;129S-*Tnf*^{tm1Gkl}

Tnfrsf1a
002818 B6.129-*Tnfrsf1a*^{tm1Mak}
003244 B6;129S-*Tnfrsf1a*^{tm1Imx} *Il1r1*^{tm1Imx}
003243 B6;129S-*Tnfrsf1a*^{tm1Imx} *Tnfrsf1b*^{tm1Imx}
003242 C57BL/6-*Tnfrsf1a*^{tm1Imx}

Tnfrsf1b
002620 B6.129-*Tnfrsf1b*^{tm1Mwm}
003243 B6;129S-*Tnfrsf1a*^{tm1Imx} *Tnfrsf1b*^{tm1Imx}
003246 B6.129S7-*Tnfrsf1b*^{tm1Imx}

Tnfrsf5
002928 B6.129P2-*Tnfrsf5*^{tm1Kik}
002927 CNcr.129P2-*Tnfrsf5*^{tm1Kik}

♦ Increased Tumor Incidence

Adenomas

JAX® GEMM® Strains

Apc^{Min} (intestinal adenomas)
002020 C57BL/6J-*Apc*^{Min}

Men1 (pancreatic b cells)
004066 FVB;129S-*Men1*^{tm1Ctre}

Prkdc^{scid} (pancreatic b cells)
002380 NOD/Lt-Tg(RipTAg)1Lt-*Prkdc*^{scid}/DvsJ

TAg (pancreatic b cells)
002033 NOD/Lt-TgN(RipTAg)1Lt

Inbred Strains

000645 A/HeJ (lung)
000646 A/J (lung)
000647 A/WySnJ (lung)

Increased Tumor Incidence cont.

Cell/Tissue Type

JAX® GEMM® Strains

Men1 (adrenal cortical tumors)
004066 FVB;129S-*Men1*^{tm1Ctre}

Gonadal Tumors

JAX® GEMM® Strains

Amh (Leydig cell tumors)
002188 B6.129S7-*Amh*^{tm1Bhr}
002187 B6;129S-*Amh*^{tm1Bhr}

BCL2
002971 FVB-TgN(BCL2OVARY)1Ah

Kit^{W} and alleles (ovarian)
000164 C57BL/6J-*Kit*^{W}
000092 FL/1Re-*Kit*^{W}
000692 WB/ReJ *Kit*^{W}/+
100410 WBB6F1/J-*Kit*^{W}/*Kit*$^{W-v}$
000350 B6By.Cg-*Mitf*$^{Mi-wh}$ *Kit*$^{W-v}$ *T*
000049 C57BL/6J-*Kit*$^{W-v}$
000194 C57BL/6J-*lx Kit*$^{W-v}$
100410 WBB6F1/J-*Kit*^{W}/*Kit*$^{W-v}$
000627 C3H/HeJ-*Kit*$^{W-x}$/+
001915 C3Hfh/HeJBm-*Kit*$^{W-x}$/+
000965 CBACa.C3-*Kit*$^{W-x}$
000133 B6.Cg-*Kit*$^{W-24J}$
000139 B6.Cg-*Kit*$^{W-25J}$
000134 C57BL/6J-*Kit*$^{W-37J}$
000847 C3Sn.B6-*Kit*$^{W-39J}$
000062 C57BL/6J-*Kit*$^{W-39J}$
000119 C57BL/6J-*Kit*$^{W-41J}$
000127 C57BL/6J-*Kit*$^{W-42J}$
001621 B6.CAST-*Gpi1a Kit*$^{W-44J}$
000122 C57BL/6J-*Kit*$^{W-44J}$
000171 B6.D2-*Kit*$^{W-45J}$
001177 B6.LP-*Kit*$^{W-49J}$
001563 B6.D2-*Kit*$^{W-73J}$

Kitl^{Sl} and alleles
000124 B6.Cg-*Ca Kitl*^{Sl} (ovarian and testicular)
000291 C3FeLe.Cg-*a/a-Ca*^{J} *Kitl*^{Sl} *Hm* (ovarian and testicular)
000693 WC/ReJ-*Kitl*^{Sl}/+ (ovarian and testicular)
100401 WCB6F1/J *Kitl*^{Sl}/*Kitl*$^{Sl-d}$ (testicular teratomas) (ovarian and testicular)
000160 B6.D2-*Kitl*$^{Sl-d}$/+ (ovarian and testicular)
000161 WB.D2-*Kitl*$^{Sl-d}$/+ (ovarian and testicular)
000090 129S1/Sv-*p*$^{+}$ *Tyr*$^{+}$ *Kitl*$^{Sl-J}$/+ (ovarian and testicular)
000979 STOCK *Kitl*$^{Sl-16J}$ (ovarian and testicular)
003252 C57BL/6J-*Kitl*$^{Sl-20J}$ (ovarian and testicular)

Men1 (ovarian and testicular)
004066 FVB;129S-*Men1*^{tm1Ctre}

Ter (testicular teratomas)
000091 129T1/Sv-+^{p} *Tyr*$^{c-ch}$ *Ter*/+

Increased Tumor Incidence cont.

Gonadal Tumors cont.

Inbred Strains

001137 129P1/ReJ (testicular teratomas)
000690 129P3/J (testicular teratomas)
002357 129P3/JEms (testicular teratomas)
001198 129P4/RrRk (testicular teratomas)
002448 129S1/SvImJ (testicular teratomas)
002064 129T2/SvEms (testicular teratomas)
002065 129T2/SvEmsJ (testicular teratomas)
000691 129X1/SvJ (testicular teratomas)
000657 CE/J (ovarian)

Recombinant Inbred Strain

001079 SWXJ-9/Bm (testicular teratomas)

Hepatomas

JAX® GEMM® Strains

Fech

002662 BALB/c-*Fech*^{m1Pas}

HBV (hepatacellular carcinoma)

002226 C57BL/6J-TgN(Alb1HBV)44Bri

Inbred Strains

000659 C3H/HeJ
000635 C3H/HeOuJ
001143 CBA/CaGnLe
000655 CBA/CaH-T6/J
000654 CBA/CaJ

Leukemia

JAX® GEMM® Strains

hr (lymphatic)

001737 B6.A-*H2-T18*^{a}.HRS-*hr*
002922 D2.HRS-*hr*
000673 HRS/J *hr*/+
001103 HRS/J-*hr Es10*^{b}/+ *Es10*^{b}
002335 SKH2/J *hr*
000147 WLHR/Le

Inbred Strains

000648 AKR/J
000668 C57L/J
000669 C58/J
000679 P/J
000680 PL/J
000682 RF/J

Lymphomas

JAX® GEMM® Strains

Atm

002753 129S6/SvEvTac-*Atm*^{tm1Awb} (thymic)

BCL2

002318 C.Cg-TgN(BCL2)22Wehi
002427 C3H/He-TgN(LCKprBCL2)36Sjk
002319 C57BL/6-TgN(BCL2)22Wehi

Increased Tumor Incidence cont.

Lymphomas cont.

JAX® GEMM® Strains

Cdc37

003690 STOCK TgN(MMTV-Cdc37)1Stp

E2f1

002785 B6;129S-*E2f1*^{tm1Meg}

HOX11

003395 CD1-TgN(Igh-HOX11)11Idd

hr (thymic)

001737 B6.A-*H2-T18*^{a}.HRS-*hr*
001103 HRS/J-*hr Es10*^{b}/+ *Es10*^{b}
002922 D2.HRS-*hr*
000673 HRS/J *hr*/+
002335 SKH2/J *hr*
000147 WLHR/Le

Myc (B cell lymphomas)

002728 C57BL/6J-TgN(IghMyc)22Bri
002677 FVB/N-TgN(WapMyc)212Bri

Prkdc^{scid} (thymic)

001303 NOD.CB17-*Prkdc*^{scid}/J
002313 NOD/LtSz-*Prkdc*^{scid} *Emv30*^{b}

Trp53

002080 129S-*Trp53*^{tm1Tyj}
002103 B6;129S-*Trp53*^{tm1Tyj}
002101 B6.129S2-*Trp53*^{tm1Tyj}
002526 C.129S2(B6)-*Trp53*^{tm1Tyj}
002547 C3Ou.129S2(B6)-*Trp53*^{tm1Tyj}
002899 FVB.129S2(B6)-*Trp53*^{tm1Tyj}
003181 FVB-TgN(MMTVneu)202Mul TgN(Trp53R172H)8512Jmr
002659 FVB/N-TgN(Trp53R172H)8512Jmr
002660 FVB/N-TgN(Trp53R172L)4491Jmr
003262 STOCK TgN(Trp53A135V)2Ber

Inbred Strains

001143 CBA/CaGnLe
000655 CBA/CaH-T6/J
000654 CBA/CaJ

Mammary Gland Tumors

JAX® GEMM® Strains

Apc^{Min}

002020 C57BL/6J-*Apc*^{Min}

Cdc37

003690 STOCK TgN(MMTV-Cdc37)1Stp

Cdh3

003180 B6.129S-*Cdh3*^{tm1Hyn}

Erbb2

002376 FVB/N-TgN(MMTVneu)202Mul

HRAS

002409 B6;SJL-TgN(WapHRAS)69Lln ^{YSJL} (males only)
002410 FVB/N-TgN(WapHRAS)69Lln ^{YSJL} (males only)

Increased Tumor Incidence cont.

Mammary Gland Tumors cont.

JAX® GEMM® Strains

MET
002675 FVB/N-TgN(MtTPRMET)243
002775 FVB/N-TgN(MtTPRMET)773

Notch4
002437 FVB/N-TgN(MMTVInt3)3Rnc
002755 FVB/N-TgN(WapInt3)10Rnc

PIP
003337 STOCK TgN(MMTVPIP)1Shu

PyVT
002374 FVB/N-TgN(MMTVPyVT)634Mul

TAg
003382 B10.D2-TgN(C3-1-TAg)cJeg
003380 C57BL/6J-TgN(C3-1-TAg)cJeg
003188 C57BL/6J-TgN(WapTAg)1Knw (males only)
003189 C57BL/6J-TgN(WapTAg)3Knw (males only)
003381 FVB-TgN(C3-1-TAg)cJeg

TGFA
002373 B6D2-TgN(MMTVTGFA)29Rjc
002459 B6D2-TgN(MMTVTGFA)254Rjc
002421 FVB/N-TgN(MtTGFA)100Lmb
002953 FVB/NJ-TgN(MMTVTGFA)254Rjc

Wnt1
002870 B6SJL-TgN(Wnt1)1Hev
002934 FVB/NJ-TgN(Wnt1)1Hev

Inbred Strains (late onset)
000645 A/HeJ
000646 A/J
000647 A/WySnJ
001026 BALB/cByJ
000651 BALB/cJ
000659 C3H/HeJ
000635 C3H/HeOuJ
001143 CBA/CaGnLe
000655 CBA/CaH-T6/J
000654 CBA/CaJ
000689 SWR/J

Other Tissues/Organs

JAX® GEMM® Strains

Blmh (multiple)
003509 B6.129-*Blmh*^{tm1Geh}

E2f1 (multiple)
002785 B6;129S-*E2f1*^{tm1Meg}

E2f1 (multiple)
002785 B6;129S-*E2f1*^{tm1Meg}

Madh3 (colorectal adenocarcinoma, metastases found in other organs)
003451 129S2/SvPasIco-*Madh3*^{tm1Par}

Increased Tumor Incidence cont.

Other Tissues/Organs cont.

JAX® GEMM® Strains

Men1 (pituitary)
004066 FVB;129S-*Men1*^{tm1Ctre}

Nf1 (multiple)
002646 B6.129S6-*Nf1*^{tm1Fcr}

Ptch (brain)
003081 B6;129-*Ptch*^{tm1Mps}

Rb1 (pituitary)
002082 129S-*Rb1*^{tm1Tyj}
002102 B6.129S2-*Rb1*^{tm1Tyj}
002548 C.129S2(B6)-*Rb1*^{tm1Tyj}
002546 C3Ou.129S2-*Rb1*^{tm1Tyj}
002900 FVB.129S2(B6)-*Rb1*^{tm1Tyj}

TAg
003445 C57BL/6J-TgN(Amy1TAg)354Knw (adipose tissue)
003446 C57BL/6J-TgN(Amy1TAg)501Knw (osteosarcoma)
002233 C57BL/6J-TgN(SV)7Bri (choroid plexus)
003477 C57BL/6J-TgN(SV)419Bri (choroid plexus)
003476 C57BL/6J-TgN(SV)427Bri (choroid plexus)

TGFA
002422 STOCK TgN(MtTGFA)42Lmb

Trp53 (osteosarcoma)
002080 129S-*Trp53*^{tm1Tyj}
002103 B6;129S-*Trp53*^{tm1Tyj}
002101 B6.129S2-*Trp53*^{tm1Tyj}
002526 C.129S2(B6)-*Trp53*^{tm1Tyj}
002547 C3Ou.129S2(B6)-*Trp53*^{tm1Tyj}
002899 FVB.129S2(B6)-*Trp53*^{tm1Tyj}
003181 FVB-TgN(MMTVneu)202Mul TgN(Trp53R172H)8512Jmr
002659 FVB/N-TgN(Trp53R172H)8512Jmr
002660 FVB/N-TgN(Trp53R172L)4491Jmr
003262 STOCK TgN(Trp53A135V)2Ber

Inbred Strains
000645 A/HeJ (lung)
000668 C57L/J (pituitary, reticulum cell neoplasm, type B)
000676 LP/J (multiple)
001902 SJL/Bm (reticulum cell sarcomas, Hodgkin's disease)
000686 SJL/J (reticulum cell sarcomas, Hodgkin's disease)
000689 SWR/J (lung)

Prostate Tumors

JAX® GEMM® Strains

Cdc37
003739 FVB.Cg-TgN(Pbsn-Cdc37)1Stp

TAg
003382 B10.D2-TgN(C3-1-TAg)cJeg
003135 C57BL/6-TgN(TRAMP)8247Ng
003380 C57BL/6J-TgN(C3-1-TAg)cJeg
003381 FVB-TgN(C3-1-TAg)cJeg

Increased Tumor Incidence cont.

Skin Cancers

JAX® GEMM® Strains

hr (Induced)
- 001737 B6.A-*H2-T18*^{a}.HRS-*hr*
- 002922 D2.HRS-*hr*
- 000673 HRS/J *hr*/+
- 001103 HRS/J-*hr Es10*^{b}/+ *Es10*^{b}
- 002335 SKH2/J *hr*
- 000147 WLHR/Le

MGMT
- 003076 NMRI/Gat-TgN(MGMT)3Bec (Resistant)

Odc
- 002647 C57BL/6-TgN(K6ODCtr)55Tgo

Tnf (Resistant)
- 003008 B6;129S-*Tnf*^{tm1Gkl}

♦ Oncogenes

JAX® GEMM® Strains

Bcl3
- 003127 129;FVB-*Bcl3*^{tm1}

Cdc37
- 003690 STOCK TgN(MMTV-Cdc37)1Stp

Erbb2
- 002376 FVB/N-TgN(MMTVneu)202Mul

Fos
- 002293 B6;129S-*Fos*^{tm1Pa}
- 002099 B6.129X1-*Fos*^{tm1Pa}

Fyn
- 002271 129-*Fyn*^{tm1Sor}
- 002385 B6;129S-*Fyn*^{tm1Sor}

HRAS
- 002409 B6;SJL-TgN(WapHRAS)69Lln Y^{SJL}
- 002410 FVB/N-TgN(WapHRAS)69Lln Y^{SJL}

Jun
- 002100 B6.129X1-*Jun*^{tm1Pa}

Kit^{W} and alleles
- 000164 C57BL/6J-*Kit*^{W}
- 000092 FL/1Re-*Kit*^{W}
- 000692 WB/ReJ *Kit*^{W}/+
- 100410 WBB6F1/J-*Kit*^{W}/*Kit*$^{W\text{-}v}$
- 000350 B6By.Cg-*Mitf*$^{Mi\text{-}wh}$ *Kit*$^{W\text{-}v}$ *T*
- 000049 C57BL/6J-*Kit*$^{W\text{-}v}$
- 000194 C57BL/6J-*lx Kit*$^{W\text{-}v}$
- 100410 WBB6F1/J-*Kit*W/*Kit*$^{tW\text{-}}$v
- 000627 C3H/HeJ-*Kit*$^{W\text{-}x}$/+
- 001915 C3Hfh/HeJBm-*Kit*$^{W\text{-}x}$/+
- 000965 CBACa.*C3*-^{Kit}W-x
- 000133 B6.Cg-*Kit*$^{W\text{-}24J}$
- 000139 B6.Cg-*Kit*$^{W\text{-}25J}$
- 000134 C57BL/6J-*Kit*$^{W\text{-}37J}$

Oncogenes cont.

JAX® GEMM® Strains

Kit^{W} and alleles cont.
- 000847 C3Sn.B6-*Kit*$^{W\text{-}39J}$
- 000062 C57BL/6J-*Kit*$^{W\text{-}39J}$
- 000119 C57BL/6J-*Kit*$^{W\text{-}41J}$
- 000127 C57BL/6J-*Kit*$^{W\text{-}42J}$
- 001621 B6.CAST-*Gpi1*^{a} *Kit*$^{W\text{-}44J}$
- 000122 C57BL/6J-*Kit*$^{W\text{-}44J}$
- 000171 B6.D2-*Kit*$^{W\text{-}45J}$
- 001177 B6.LP-*Kit*$^{W\text{-}49J}$
- 001563 B6.D2-*Kit*$^{W\text{-}73J}$

Kras2
- 002674 129-*Kras2*^{tm1Tyj}

luc
- 003479 B6.C3-Tg(Fos-luc)1Rnd

Mos
- 002722 129S6/SvEv-*Mos*^{tm1Ev}
- 002723 B6.129S6-*Mos*^{tm1Ev}
- 002404 STOCK *Mos*^{tm1Ev}

Myc
- 002728 C57BL/6J-TgN(IghMyc)22Bri
- 002677 FVB/N-TgN(WapMyc)212Bri

MYC/ESR
- 002712 FVB/N-TgN(PF4MER)6Kra

Rab3a
- 002443 B6;129S-*Rab3a*^{tm1Sud}

Rela
- 002851 B6;129S-*Rela*^{tm1Bal}

Shh
- 003318 STOCK *Shh*^{tm1Amc}

Src
- 002278 129-*Src*^{tm1Sor}
- 002381 B6;129S-*Src*^{tm1Sor}
- 002277 B6.129S7-*Src*^{tm1Sor}

Yes
- 002280 129-*Yes*^{tm1Sor}

♦ Other

JAX® GEMM® Strains

Aprt (DNA Repair)
- 002779 129S-*Aprt*^{tm1Zqw}

Cd44 (tumor mestastasis)
- 003899 B6;129-*Cd44*^{tm1Hbg}

FCGR2A
- 003542 B6;SJL-TgN(FCGR2A)11Mkz

Plg
- 002830 B6.129P2-*Plg*^{tm1Jld}

Other cont.

JAX® GEMM® Strains

TAg (tumor mestastasis)
003445 C57BL/6J-TgN(Amy1TAg)354Knw
003446 C57BL/6J-TgN(Amy1TAg)501Knw

Terc (DNA Repair)
004132 B6.Cg-*Terc*^{tm1Rdp}

♦ Research Tools

JAX® GEMM® Strains

BCL2
002319 C57BL/6-TgN(BCL2)22Wehi
002320 C57BL/6-TgN(BCL2)25Wehi
002321 C57BL/6-TgN(BCL2)36Wehi
002318 C.Cg-TgN(BCL2)22Wehi

Btk (B cell deficiency)
002536 B6;129S-*Btk*^{tm1Wk}

Cd44 (tumor immunology)
003899 B6;129-*Cd44*^{tm1Hbg}

Epas1 (endothelial cell marker for neovascularization)
003266 B6;129-*Epas1*^{tm1Rus}

GFP
003658 FVB/N-TgN(TIE2GFP)287Sato

Igh-6 (B cell deficiency)
003751 B6;129S-*Igh-6*^{tm1Che}
002288 B6.129S2-*Igh-6*^{tm1Cgn}
002249 B10.129S2(B6)-*Igh-6*^{tm1Cgn}
003903 NOD.129S2-*Igh-6*^{tm1Cgn}/Dvs
002572 NOD.129S2(B6)-*Igh-6*^{tm1Cgn}

Jak3 (B, T, and NK cell deficiency) (xenograft/transplant host)
002852 B6.129S4-*Jak3*^{tm1Ljb}

lacZ
002754 C57BL/6-TgN(LacZpl)60Vij
002856 FVB/N-TgN(TIE2LacZ)182Sato

MCL1
004187 B6;SJL-Tg(MCL1)8Caig

MGMT
003076 NMRI/Gat-TgN(MGMT)3Bec

Prkdc^{scid} (B & T cell deficiency) (xenograft/transplant host)
001913 B6.CB17-*Prkdc*^{scid}/SzJ
002577 B6;CB17-*Ghrhr*^{lit} *Prkdc*^{scid}/Bm
001131 C3Smn.CB17-*Prkdc*^{scid}/J
002038 CB17;HPG-*Prkdc*^{scid} *Gnrh*^{hpg}/Bm
001803 CBySmn.CB17-*Prkdc*^{scid}/J
001303 NOD.CB17-*Prkdc*^{scid}/J)
002570 NOD.Cg-*Prkdc*^{scid} *B2m*^{tm1Unc}/J
002579 NOD.Cg-*Prkdc*^{scid}-TgN(L-FABP, S-GH)7Bir/Bm
003843 NOD/Lt-*Prkdc*^{scid} Tg(GAD2)1Lt
003844 NOD/Lt-*Prkdc*^{scid} Tg(GAD2)2Lt
002380 NOD/Lt-Tg(RipTAg)1Lt-*Prkdc*^{scid}/DvsJ
002313 NOD/LtSz-*Prkdc*^{scid} *Emv30*^{b}
003449 NOD/LtSz-*Prkdc*^{scid} *B2m*^{tm1Unc}

Research Tools cont.

JAX® GEMM® Strains

Rac2 (B cell deficiency) (T cell deficiency) (production of B cells and antibodies)
004197 B6.129S6-*Rac*^{2tm1Mddw}

Rag1 (B & T cell deficiency) (xenograft/transplant host)
002096 B6;129S-*Rag1*^{tm1Mom}
002216 B6.129S7-*Rag1*^{tm1Mom}
003145 C.129S7(B6)-*Rag1*^{tm1Mom}
003729 NOD.129S7(B6)-*Rag1*^{tm1Mom}/J
002506 STOCK TgN(CD3E)26Cpt-*Rag1*^{tm1Mom}

Tcra (specific T cell deficiency)
002115 B6;129S-*Tcra*^{tm1Mom}
002116 B6.129S2-*Tcra*^{tm1Mom}
002761 B10.Cg-TgN(TcrAND)53Hed
003147 B10.D2-*H2*^{d} *H2-T18*^{c} *Hc*^{1}/nSnJ-TgN(DO11.10)10Loh
003199 B10.PL-*H2*^{u} *H2-T18*^{a}(73NS)/Sn-TgN(TCRA)B1Jg
002045 C.SJL-*Tcra*^{c}/Slk

Tcra Tcrb (specific T cell deficiency)
002331 B6;D2-TgN(TcrLCMV)327Sdz
002408 B6;SJL-TgN(TcrAND)53Hed
003303 BALB/c-TgN(DO11.10)10Loh
002047 C.SJL-*Tcra*^{a} *Tcrb*^{c}/Slk
003831 C57BL/6-Tg(TcraTcrb)1100Mjb
002597 STOCK TgN(TcrHEL3A9)Mmd

Tcrb (specific T cell deficiency)
002117 B6;129P-*Tcrb*^{tm1Mom}
002118 B6.129P2-*Tcrb*^{tm1Mom}
002761 B10.Cg-TgN(TcrAND)53Hed
003147 B10.D2-*H2*^{d} *H2-T18*^{c} *Hc*^{1}/nSnJ-TgN(DO11.10)10Loh
003200 B10.PL-*H2*^{u}*H2-T18*^{a}(73NS)/Sn-TgN(TCRB)C14Jg
002046 C.SJL-*Tcrb*^{a}/Slk
003540 C57L/J-TgN(Tcrb)93Vbo

Tcrb Tcrd (specific T cell deficiency)
002121 B6;129P-*Tcrb*^{tm1Mom} *Tcrd*^{tm1Mom}
002122 B6.129P2-*Tcrb*^{tm1Mom} *Tcrd*^{tm1Mom}

Tcrd (specific T cell deficiency)
002119 B6;129P-*Tcrd*^{tm1Mom}
002120 B6.129P2-*Tcrd*^{tm1Mom}

tTA
002618 C57BL/6J-TgN(MMTVtTA)1Mam

Inbred Strains

000646 A/J
000649 AU/SsJ
002846 BALB/cAnLil
001026 BALB/cByJ
001905 BALB/cGa
000921 BALB/cGrRk
000651 BALB/cJ
001311 BALB/cWtEi
000653 BUB/BnJ (no detectable endogenous ecotropic MuLV DNA Sequences)
003719 MSM/Ms
000682 RF/J

Research Tools cont.

Inbred Strains cont.
000683 RIIIS/J

Recombinant Inbred Strains
AKXD RI Lines
AKXL RI Lines
CX8 RI Lines
SWXJ RI Lines

Miscellaneus Strains
001802 CBy.RBF-Rb5Bnr/J
000726 RBF/DnJ

♦ Toxicology

JAX® GEMM® Strains

Abcb4
002539 FVB.129P2-*Abcb4*^{tm1Bor}

Ahr
002831 B6.129-*Ahr*^{tm1Bra}
002727 B6;129S-*Ahr*^{tm1Bra}

Blmh
003509 B6.129-*Blmh*^{tm1Geh}

hr
001737 B6.A-*H2-T18*^{a}.HRS-*hr*
002922 D2.HRS-*hr*
000673 HRS/J *hr*/+
001103 HRS/J-*hr Es10*^{b}/+ *Es10*^{b}
002335 SKH2/J *hr*
000147 WLHR/Le

lacZ
002754 C57BL/6-TgN(LacZpl)60Vij

Odc
002647 C57BL/6-TgN(K6ODCtr)55Tgo

Rag1 (B & T cell deficiency) (xenograft/transplant host)
002096 B6;129S-*Rag1*^{tm1Mom}
002216 B6.129S7-*Rag1*^{tm1Mom}
003145 C.129S7(B6)-*Rag1*^{tm1Mom}
003729 NOD.129S7(B6)-*Rag1*^{tm1Mom}/J
002506 STOCK TgN(CD3E)26Cpt-*Rag1*^{tm1Mom}

Trp53
002080 129S-*Trp53*^{tm1Tyj}
002103 B6;129S-*Trp53*^{tm1Tyj}
002101 B6.129S2-*Trp53*^{tm1Tyj}
002526 C.129S2(B6)-*Trp53*^{tm1Tyj}
002547 C3Ou.129S2(B6)-*Trp53*^{tm1Tyj}
003181 FVB-TgN(MMTVneu)202Mul TgN(Trp53R172H)8512Jmr
002899 FVB.129S2(B6)-*Trp53*^{tm1Tyj}
002659 FVB/N-TgN(Trp53R172H)8512Jmr
002660 FVB/N-TgN(Trp53R172L)4491Jmr
003262 STOCK TgN(Trp53A135V)2Ber

Inbred Strains
002747 SENCARB/PtJ
002748 SENCARC/PtJ
002931 SSIN/Sprd

♦ Tumor Suppressor Genes

JAX® GEMM® Strains

Atm
002753 129S6/SvEvTac-*Atm*^{tm1Awb}

Bcl2
003082 129S1/Sv-*Bcl2*^{tm1Mpin}
002265 B6;129S-*Bcl2*^{tm1Sjk}

BCL2
002318 C.Cg-TgN(BCL2)22Wehi
002319 C57BL/6-TgN(BCL2)22Wehi

Blmh
003509 B6.129-*Blmh*^{tm1Geh}

Cdkn1a
003263 B6;129-*Cdkn1a*^{tm1Tyj}

E2f1
002785 B6;129S-*E2f1*^{tm1Meg}

Men1
004066 FVB;129S-*Men1*^{tm1Ctre}

Odc
002647 C57BL/6-TgN(K6ODCtr)55Tgo

Ptch
003081 B6;129-*Ptch*^{tm1Mps}

Rb1 (pituitary tumors)
002082 129S-*Rb1*^{tm1Tyj}
002102 B6.129S2-*Rb1*^{tm1Tyj}
002548 C.129S2(B6)-*Rb1*^{tm1Tyj}
002546 C3Ou.129S2-*Rb1*^{tm1Tyj}
002900 FVB.129S2(B6)-*Rb1*^{tm1Tyj}

Terc
004132 B6.Cg-*Terc*^{tm1Rdp}

Trp53
002080 129S-*Trp53*^{tm1Tyj}
002103 B6;129S-*Trp53*^{tm1Tyj}
002101 B6.129S2-*Trp53*^{tm1Tyj}
002526 C.129S2(B6)-*Trp53*^{tm1Tyj}
002547 C3Ou.129S2(B6)-*Trp53*^{tm1Tyj}
003181 FVB-TgN(MMTVneu)202Mul TgN(Trp53R172H)8512Jmr
002899 FVB.129S2(B6)-*Trp53*^{tm1Tyj}
002659 FVB/N-TgN(Trp53R172H)8512Jmr
002660 FVB/N-TgN(Trp53R172L)4491Jmr
003262 STOCK TgN(Trp53A135V)2Ber

Ttpa
003823 B6.129S4-*Ttpa*^{tm1Far}

Vhlh
003123 129S;ICR-*Vhlh*^{tm1Big}
004081 C;129S-*Vhlh*^{tm1Jae}

Wt1
002332 B6;129S-*Wt1*^{tm1Jae}
002719 B6.129S4-*Wt1*^{tm1Jae}

INBRED STRAINS

Strain Name **129P3/J**
Former & Common Name(s): 129/J; 129P3
Stock Number **000690**
Application(s) Cancer Research: Increased Tumor Incidence (Gonadal Tumors, testicular teratomas)
Additional Research Areas
Cardiovascular Research; Neurobiology Research; Reproductive Biology Research; Sensorineural Research; Research Tools: General Purpose, Genetics Research

Strain Name **129S1/SvImJ**
Former & Common Name(s): 129S3/SvImJ; 129/SvImJ
Stock Number **002448**
Application(s) Cancer Research: Increased Tumor Incidence (Gonadal Tumors, testicular teratomas)
Additional Research Areas
Neurobiology Research; Reproductive Biology Research; Sensorineural Research; Research Tools: General Purpose; Genetics Research

Strain Name **129T2/SvEmsJ**
Former & Common Name(s): 129/SvEms-+^{Ter} ?/J; 129T2
Stock Number **002065**
Application(s) Cancer Research: Increased Tumor Incidence (Gonadal Tumors, testicular teratomas)
Additional Research Areas
Reproductive Biology Research; Research Tools: Genetics Research

Strain Name **129X1/SvJ**
Former & Common Name(s): 129/SvJ; 129X1
Stock Number **000691**
Application(s) Cancer Research: Increased Tumor Incidence (Gonadal Tumors, testicular teratomas)
Additional Research Areas
Neurobiology Research; Reproductive Biology Research; Sensorineural Research; Research Tools: General Purpose; Genetics Research

Phenotype For a complete history of the numerous 129 substrains please refer to Simpson, *et al.*, 1997. Historically, the 129 inbred mice are known for the high incidence of spontaneous testicular teratomas, though the incidence differs between substrains. Most recently 129 mice are widely used strain in the production of targeted mutations due to the availability of several lines of embryonic stem cells. There is major genetic variation within the 129 "family", which has led to an update of the nomenclature and a division of substrains into three major groups: parental substrains, steel substrains and "ter" substrains. Investigators using 129 substrains for targeted mutagenesis should be careful in the selection of the appropriate 129 substrain to match the embryonic stem cell line.

Selected References

Festing MF, Simpson EM, Davisson MT, Mobraaten LE. 1999. Revised nomenclature for strain 129 mice. *Mamm Genome* 10:836-7.

Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).

Simpson EM, Linder CC, Sargent EE, Davisson MT, Mobraaten LE, Sharp JJ. 1997. Genetic variation among 129 substrains and its importance for targeted mutagenesis in mice. *Nat Genet* 16:19-27.

Stevens LC. 1973. A new inbred subline of mice (129/ter, Sv) with a high incidence of spontaneous congenital testicular teratomas. *J Natl Cancer Inst* 50:235-42.

Threadgill DW, Yee D, Matin A, Nadeau JH, Magnuson T. 1997. Genealogy of the 129 inbred strains 129Sv/J is a contaminated inbred strain. *Mamm Genome* 8:390-3.

FEATURED MODELS

Strain Name **A/J**

Stock Number **000646**

Application(s) Cancer Research: Increased Tumor Incidence (Adenomas, lung) (Mammary Gland Tumors, late onset)

Additional Research Areas

Cardiovascular Research; Developmental Biology Research; Sensorineural Research; Research Tools: Cancer Research; General Purpose; Immunology and Inflammation Research

Phenotype Developed by LC Strong in 1921 from a cross between a Cold Spring Harbor albino and a Bagg albino, the A inbred strain is used widely used in cancer and immunology research. It is highly susceptible to induction of congenital cleft palate by cortisone. It has a high incidence of spontaneous lung adenomas and lung tumors readily develop in response to carcinogens. High percentage of mammary adenocarcinomas (a large proportion acinar type) develop in multiparous females. Rare spontaneous myoepitheliomas arising from myoepithelial cells of various exocrine glands have been observed in The Jackson Laboratory substrains. A/J mice, fed an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat), fail to develop atherosclerotic aortic lesions in contrast to several highly susceptible strains of mice (e.g. C57BL/6J, Stock No. 000664; C57L/J, Stock No. 000668, C57BR/cdJ, Stock No. 000667, and SM/J, Stock No. 000687).

Selected References

Heston WE. 1963. Genetics of neoplasia. In: *Methodology in Mammalian Genetics*, Burdette WJ, (ed), Holden-Day, San Francisco, pp. 247-68.

Hoag WG. 1963. Spontaneous cancer in mice. *Ann NY Acad Sci* 108:805-31.

Sundberg JP, Hanson CA, Roopenian DR, Brown KS, Bedigian HG. 1991. Myoepitheliomas in inbred laboratory mice. *Vet Pathol* 28:313-23.

Strain Name **AKR/J**

Former & Common Name(s): Acact^{ald}; ald

Stock Number **000648**

Application(s) Cancer Research: Increased Tumor Incidence (Leukemia, lymphatic)

Additional Research Areas

Cardiovascular Research; Endocrine Deficiency Research; Internal/Organ Research; Metabolism Research

Phenotype Originally inbred at the Rockefeller Institute, AKR mice are widely used in cancer research for their high leukaemia incidence (60-90%) and in immunology as a source of the Thy1.1 (theta AKR) antigen. Mice of this strain are viremic from birth and express in all tissues the ecotropic retrovirus AKV.

Selected References

Festing MFW, Blackmore DK. 1971. Life span of specified-pathogen-free (MRC category 4) mice and rats. *Lab Anim* 5:179-92.

Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).

Lilly F, Pincus T. 1973. Genetic control of murine viral leukemogenesis. *Adv Cancer Res* 17:231-77.

Nemirovsky T, Trainin N. 1973. Leukemia induction in C3H mice following their inoculation with normal AKR lymphoid cells. *Int J Cancer* 11:172-7.

Strain Name **BALB/cByJ**

Former & Common Name(s): BALB Bailey; CBy

Stock Number **001026**

Application(s) Cancer Research: Increased Tumor Incidence (Mammary Gland Tumors, late onset)

Additional Research Areas

Neurobiology Research; Research Tools: Cancer Research; General Purpose; Immunology and Inflammation Research

Strain Name **BALB/cJ**
Former & Common Name(s): BALB; C
Stock Number **000651**
Application(s) Cancer Research: Increased Tumor Incidence (Mammary Gland Tumors, late onset)
Additional Research Areas
Cardiovascular Research; Neurobiology Research; Research Tools: General Purpose; Immunology and Inflammation Research

Phenotype BALB/c mice are particularly well known for the production of plasmacytomas on injection with mineral oil forming the basis for the production of monoclonal antibodies. Mammary tumor incidence is normally low but infection with mammary tumor virus by fostering to MMTV$^+$ C3H mice dramatically increases tumor number and age of onset. BALB/c mice develop other cancers later in life including reticular neoplasms, primary lung tumors, and renal tumors. Rare spontaneous myoepitheliomas arising from myoepithelial cells of various exocrine glands have been observed in both BALB/cJ and BALB/cByJ substrains.

Selected References

Bentvelzen P, Daams JH, Hageman P, Calafat J. 1970. Genetic transmission of viruses that incite mammary tumors in mice. *Proc Natl Acad Sci USA* 67:377-84.

Ebbesen P. 1971. Reticulosarcoma and amyloid development in BALB/c mice inoculated with syngeneic cells from young and old donors. *J Natl Cancer Inst* 47:1241-5.

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Sass B, Peters RL, Kelloff GJ. 1976. Differences in tumor incidence in two substrains of claude BALB/c (BALB/CfCd) mice, emphasizing renal, mammary, pancreatic and synovial tumors. *Lab Anim Sci* 26:736-41.

Schlom J, Michalides R, Kufe D, Hehlmann R, Spiegelman S, Bentvelzen P, Hageman P. 1973. A comparative study of the biological and molecular basis of murine mammary carcinoma. A model for human breast cancer. *J Natl Cancer Inst* 51:541-51.

Sundberg JP, Hanson CA, Roopenian DR, Brown KS, Bedigian HG. 1991. Myoepitheliomas in inbred laboratory mice. *Vet Pathol* 28:313-23.

Strain Name **C3H/HeJ**
Former & Common Name(s): C3H/HeJ MMTV$^-$; C3; C3H Heston; *Lps^d;Pd eb^{rd1};rd 1*
Stock Number **000659**

Phenotype C3H/HeJ mice are used as a general purpose strain in a wide variety of research areas including cancer, immunology and inflammation, sensorineural, and cardiovascular biology research. C3H/HeJ mice and all other Jackson substrains are homozygous for the retinal degeneration 1 mutation (*Pdeb^{rd1}*) causing blindness by weaning age. There is also a high incidence of hepatomas in C3H mice (reportedly 72-91% in males at 14 months, 59% in virgin females, 30-38% in breeding females). Despite the lack of exogenous mouse mammary tumor virus (*MMTV*), virgin and breeding females may still develop some mammary tumors later in life. C3H/HeJ mice, fed an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat), fail to develop atherosclerotic aortic lesions in contrast to several highly susceptible strains of mice (e.g. C57BL/6J, Stock No. 000664; C57L/J, Stock No. 000668, C57BR/cdJ, Stock No. 000667, and SM/J, Stock No. 000687).

FEATURED MODELS

Strain Name **C3H/HeOuJ**

Former & Common Name(s): C3H/HeOuJ MMTV-; C3H Outzen; C3Ou; *Pdeb^{rd1};rd 1*

Stock Number **000635**

Application(s) Cancer Research: Increased Tumor Incidence (Hepatomas) (Mammary Gland Tumors, late onset)

Additional Research Areas

Mouse Models for Human Disease; Sensorineural Research; Research Tools: General Purpose

000659 only

Cardiovascular Research; Immunology and Inflammation Research;

Phenotype C3H/HeOuJ mice are used as a general purpose strain in a wide variety of research areas including cancer and sensorineural, research. C3H/HeOuJ mice and all other C3H substrains at The Jackson Laboratory are homozygous for the retinal degeneration 1 mutation (*Pdeb^{rd1}*), causing blindness by weaning age. There is also a high incidence of hepatomas in C3H mice (reportedly 72-91% in males at 14 months, 59% in virgin females, 30-38% in breeding females). Despite the lack of exogenous mouse mammary tumor virus (*MMTV*), virgin and breeding females may still develop some mammary tumors later in life.

Selected References

Dragani TA, Manenti G, Gariboldi M, De Gregorio L, Pierotti MA. 1995. Genetics of liver tumor susceptibility in mice. *Toxicol Lett* 82-83:613-9.

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Heston WE. 1963. Genetics of neoplasia. In: *Methodology in Mammalian Genetics*, Burdette WJ, (ed), Holden-Day, San Francisco, pp. 247-68.

Heston WE, Vlahakis G. 1971. Mammary tumours, plaques and hyperplastic alveolar nodules in various combinations of mouse inbred strains and the different lines of the mammary tumour virus. *Int J Cancer* 7:141-8.

Outzen HC, Corrow D, Shultz LD. 1985. Attenuation of exogenous murine mammary tumor virus virulence in the C3H/HeJ mouse substrain bearing the Lps mutation. *J Natl Cancer Inst* 75:917-23.

Strain Name **C57L/J**

Former & Common Name(s): C57 leaden

Stock Number **000668**

Application(s) Cancer Research: Increased Tumor Incidence (Leukemia) (Other Tissues/Organs, pituitary, reticulum cell neoplasm, type B)

Additional Research Areas

Cardiovascular Research, Dermatology Research; Immunology and Inflammation Research; Neurobiology Research; Sensorineural Research; Research Tools: General Purpose

Phenotype C57L/J mice are used widely in research as a general purpose strain. Mice have a high incidence of Hodgkin's-like reticulum cell neoplasm at 18 months of age and pituitary tumors in old multiparous females. C57L/J mice are highly susceptible to experimental allergic encephalomyelitis (EAE). In addition, C57L/J mice are highly susceptible to developing atherosclerotic aortic lesions (4500 to 8000 um^2 atherosclerotic aortic lesions/aortic cross-section) following 14 weeks on an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat). C57L/J mice carry no detectable endogenous ecotropic MuLV DNA sequences.

Selected References

Dunn TB, Deringer MK. 1968. Reticulum cell neoplasm, type B, or the "Hodgkin's-like lesion" of the mouse. *J Natl Cancer Inst* 40:771-821.

Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).

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Hoag WG. 1963. Spontaneous cancer in mice. *Ann NY Acad Sci* 108:805-31.

Jenkins NA, Copeland NG, Taylor BA, Lee BK. 1982. Organization, distribution, and stability of endogenous ecotropic murine leukemia virus DNA sequences in chromosomes of Mus musculus. *J Virol* 43:26-36.

Murphy ED. 1966. Characteristic tumors. In: *Biology of the Laboratory Mouse*, 2nd Edition, Green EL, (ed), McGraw-Hill, New York, pp. 521-62.

Strain Name **CBA/CaJ**
Former & Common Name(s): CBA Carter J
Stock Number **000654**
Application(s) Cancer Research: Increased Tumor Incidence (Hepatomas) (Lymphomas) (Mammary Gland Tumors, late onset)
Additional Research Areas
Diabetes and Obesity Research; Reproductive Biology Research; Research Tools: General Purpose

Phenotype The CBA inbred strain was initially bred for longevity and low tumor incidence. Burdette and Strong reported that CBA mice were comparatively susceptible to tumor induction after a single subcutaneous injection of methylcholanthrene. The tumor types identified in this early work in CBA mice included spindle cell sarcoma, rhabdomyosarcoma, and epidermoid carcinoma. Strong and Smith reported finding benign hepatomas in aging CBA mice. Several groups confirmed this finding and the majority of studies found a higher frequency of spontaneous hepatomas in males than in females.
CBA/Ca mice are commonly used for leukemogenesis research since this strain has a low spontaneous incidence of leukemia but has a relatively high inducibility of myeloid leukemia in response to benzene and radiation exposure. Multiple reports using CBA, its F1 hybrids, and other strains, have indicated that deletions in a specific segment of chromosome 2 are linked to radiation and chemical induction of myeloid leukemia. This segment is reported to map to a 1 cM interval flanked by D2Mit126 and D2Mit185 which is homologous to human chromosome segment 11p11-12.
In addition, CBA/Ca mice have been used for the assessment of cytostatic drug combination protocols and have also been utilized successfully as hosts for childhood rhabdomyosarcoma xenografts, after thymectomy and irradiation. CBA/CaJ mice carry viral proteins Mtv8, Mtv9, and Mtv14.

Selected References
Rithidech KN, Cronkite EP, Bond VP. 1999. Advantages of the CBA mouse in leukemogenesis research. *Blood Cells Mol Dis* 25:38-45.
Strong LC. 1936. Production of the CBA strain of inbred mice: long life associated with low tumour incidence. *Brit J Exp Path* 17:60-3.

Strain Name **PL/J**
Former & Common Name(s): *Pdeb*^{rd1}; *rd1*
Stock Number **000680**
Application(s) Cancer Research: Increased Tumor Incidence (Leukemia)
Additional Research Areas
Immunology and Inflammation Research; Mouse Models for Human Disease; Sensorineural Research

Phenotype PL/J mice show a moderate susceptibility to experimental allergic encephalitis with late onset and high mortality. Reports of leukemia incidence vary from 50% in females and 19% in males to 80-90%.

Selected References
Albert S, Wolf PL, Pryjma I, Moore W. 1965. Thymus development in high and low-leukemic mice. *J Reticuloendothel Soc* 2:218-37.
Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).
Heston WE. 1968. Genetic aspects of experimental animals in cancer research. *Jap Cancer Assoc Gann Monograph* 5:3-15.

Strain Name **RBF/DnJ**
Stock Number **000726**
Application(s) Research Tools: Cancer Research (myeloma and hybridoma production)
Additional Research Areas
Research Tools: Genetics Research, Sensorineural Research

Phenotype The RBF inbred strain arose from crosses with wild mice, originally known as "tobacco mouse", captured in Valle di Poschiavo in S.E. Switzerland. The wild mice originally

FEATURED MODELS

known as 'tobacco mouse' because of the coat colour. The strain was transferred to Dr. M. Davisson (Dn) in 1981 and subsequently to the production colony of The Jackson Laborotory (J). Mice are homozygous for Robertosonian translocation Rb(1.3)1Bnr, Rb(8.12)5Bnr and Rb(9.14)6Bnr. This strain is useful for production of antibody producing hybridomas.

Selected References

Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).
Taggart RT, Samloff IM. 1983. Stable antibody-producing murine hybridomas. *Science* 219:1228-30.
Robertsonian Chromosome Resource. http://jaxmice.jax.org/html/jaxnotes/jaxn434a.shtml. *JAX Notes* 1988:434 July.

Strain Name **SJL/J**

Stock Number **000686**

Application(s)

Cancer Research: Increased Tumor Incidence (Other Tissues/Organs, reticulum cell sarcomas, Hodgkin's disease)

Additional Research Areas

Cardiovascular Research; Diabetes and Obesity Research; Immunology and Inflammation Research; Mouse Models for Human Disease; Sensorineural Research

Phenotype

SJL mice display a very high incidence of reticulum cell sarcomas resembling Hodgkin's disease around one year of age. Sarcomas first appear in the Peyer's patches and mesenteric lymph nodes and later in the spleen, liver, thymus and other lymph nodes. Most of the tumors are mixed-cell types classified as type B reticulum cell neoplasms, but a few are type A histiocytomas. This strain is also characterized by extreme agression in males and its susceptibility to experimental autoimmune encephalomyelitis (EAE) for multiple sclerosis research. SJL/J mice develop a spontaneous myopathy resulting from a splice-site mutation in the *Dysferlin* gene. This $Dysf^{lm}$ allele has been shown to result in decreased levels of dysferlin protein in SJL/J mice and makes this strain a good model for limb girdle muscular dystrophy 2B. This spontaneous myopathy is characterized by a progressive loss of muscle mass and strength corresponding with an increase in muscle pathology including muscle fibers with central nuclei, variation in size, splitting, inflammatory infiltrate, necrosis, and eventual replacement of muscle fiber with fat. While muscle weakness can be detected as early as three weeks of age the greatest pathology occurs after 6 months of age. SJL/J mice have also been shown to have an increased rate of muscle regeneration after injury when compared to BALB/c mice.

SJL mice, fed an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat), fail to develop atherosclerotic aortic lesions in co ntrast to several highly susceptible strains of mice (*e.g.* C57BL/6J, Stock No. 000664; C57L/J, Stock No. 000668, C57BR/cdJ, Stock No. 000667, and SM/J, Stock No. 000687). SJL/J are also useful as a control strain for studying immune defects in NOD/LtJ mice (Stock No. 001976), a model for type I diabetes (IDDM). Both NOD and SJL/J are derived from swiss mice; SJL are immunocompetent but have elevated levels of circulating T cells.

Selected References

Crispens CG. 1973. Some characteristics of strain SJL/JDg mice. *Lab Anim Sci* 23:408-13.
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Fujinaga S, Poel WE, Williams WC, Dmochowski L. 1970. Biological and morphological studies of SJL/J strain reticulum cell neoplasms induced and transmitted serially in low leukemia-strain mice. *Cancer Res* 30:729-42.
Leiter EH. 1998. NOD mice and related strains: origins, husbandry and biology introduction. In: *NOD Mice and Related Strains: Research Applications in Diabetes, AIDS, Cancer and Other Diseases*. Leiter EH, Atkinson MA (eds), RG Landes, Austin, pp. 1-23.
Murphy ED. 1963. SJL/J, a new inbred strain of mouse with a high, early incidence of reticulum-cell neoplasms. *Proc Am Assoc Cancer Res* 4:46.

Strain Name **SWR/J**

Former & Common Name(s):L y-24; Ly24; Pgp-1; Pgp1

Stock Number **000689**

Application(s) Cancer Research: Increased Tumor Incidence (Mammary Gland Tumors) (Other Tissues/ Organs, lung)

Additional Research Areas

Cardiovascular Research; Diabetes and Obesity Research; Immunology and Inflammation Research; Mouse Models for Human Disease; Research Tools: General Purpose, Sensorineural Research

Phenotype SWR/J mice are used widely in research as a general purpose strain. Aging mice exhibit a high incidence of lung and mammary gland tumors. They also develop extreme polydipsia and polyuria (nephrogenic diabetes insipidus) with increasing age. SWR/J mice are highly susceptible to experimental allergic encephalomyelitis (EAE). They are resistant to collagen-induced arthritis. SWR/J mice show an intermediate susceptibility to developing atherosclerotic aortic lesions (1670 to 1690 μ^2 atherosclerotic aortic lesions/aortic cross-section) following 14 weeks on an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat). SWR/J mice have been recommended for generation and propogation of transgenic mice because they are high responders to exogenous hormones, have large and prominant pronuclei with good resistance to lysis following microinjection, and are genetically well-defined. SWR/J mice may also be used as controls for comparison to the autoimmune diabetic NOD/LtJ mice (Stock No. 001976), especially for experiments examining the abberant immune functions of NOD/LtJ mice. Both NOD and SWR/J mice are derived from swiss mice. SWR/J are in some cases more suitable than random bred swiss ICR mice because of their genetic uniformity. Unlike NOD/LtJ mice they are not immunocompromised, and they are genetically very different from NOD.

Selected References

Deringer MK 1970. Mammary tumors in strains BL/LyDe and SWR/LyDe mice. *J Natl Cancer Inst* 45:215-18.

Festing MFW. 1999. Inbred strains of mice. Mouse Genome Informatics, The Jackson Laboratory, Bar Harbor, Maine. World Wide Web (URL: http://www.informatics.jax.org/).

Heston WE. 1963. Genetics of neoplasia. In: *Methodology in Mammalian Genetics*, Burdette WJ, (ed), Holden-Day, San Francisco, pp. 247-68.

FEATURED MODELS

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Symbol
Cdkn1b

Gene Name **cyclin-dependent kinase inhibitor 1B (P27)**

p27^{kip}, encoded by *Cdkn1b*, is a member of the Kip/Cip family of cyclin-dependent kinase inhibitors. In humans, recent reports indicate that reduced expression of *CDKN1B* in primary breast carcinomas is independently prognostic for reduced disease-free survival, particularly in younger women, in whom *CDKN1B* expression was also prognostic for reduced overall survival. Thus, these mice will be useful for examining the involvement of *Cdkn1b* on mammary gland development, differentiation, and carcinogenesis.

Strain Name **129/Sv-*Cdkn1b*^{tm1Mlf}**
Stock Number **003122**
Licensing OncoMouse™; requires license from DuPont.
Use of these mice for commercial purposes or by a for-profit entity will require a license from the originating institution; please inquire.
Application(s) Cancer Research: Genes Regulating Growth and Proliferation
Additional Research Areas
Cell Biology Research; Reproductive Biology Research
Phenotype Mice deficient in p27^{kip} are viable and larger than normal littermates, with increased cellularity of all tissues. The thymus and spleen are particularly enlarged. Nullizygous adult mice have a shortened lifespan due to the growth of benign intermediate lobe pituitary tumors. Female mice are infertile, with a follicular phase ovulatory block. Large doses of exogenous gonadotropin induce ovulation, but both implantation and intrauterine embryonic development is impaired. The mice demonstrate haploid-insufficient susceptibility to the development of adenomas in the pituitary, intestine and lung adenomas following exposure to gamma irradiation or chemical carcinogens.
Primary Reference Fero ML, Rivkin M, Tasch M, Porter P, Carow CE, Firpo E, Polyak K, Tsai L-H, Broudy V, Perlmutter RM, Kaushansky K, Roberts JM. 1996. A syndrome of multiorgan hyperplasia with features of gigantism, tumorigenesis, and female sterility in p27Kip1-deficient mice. *Cell* 85:733-44.

Symbol
Erbb2

Gene Name **avian erythroblastosis oncogene B-2**

Strain Name **FVB/N-TgN(MMTVneu)202Mul**
Former & Common Name(s):MMT V neu
Promoter:MMT V, mouse mammary tumor virus
Stock Number **002376**
Licensing OncoMouse™; requires license from DuPont.
Use of these mice for commercial purposes or by a for-profit entity will require a license from the originating institution; please inquire.
Application(s) Cancer Research: Increased Tumor Incidence (Mammary Gland Tumors); Oncogenes
Phenotype Mice homozygous for the MMTVneu (rat) transgene are viable and fertile. There is no phenotypic effect in males. The transgene is expressed at low levels in normal mammary epithelium, salivary gland and lung. Higher expression was detected in tumor tissue. Focal mammary tumors first appear at 4 months, with a median incidence of 205 days. Both virgin and breeder mice develop tumors. Tumors arose as foci in hyperplastic, dysplastic mammary glands. Seventy-two percent of tumor-bearing mice that lived to 8 months or longer developed metastatic disease to the lung. The phenotype of MMTV/unactivated neu transgenic mice differs from that of the MMTV/activated neu produced by Phil Leder, in which multifocal tumors involving the entire mammary epithelium arise.

Primary Reference Guy CT, Webster MA, Schaller M, Parsons TJ, Cardiff RD, Muller WJ. 1992. Expression of the neu protooncogene in the mammary epithelium of transgenic mice induces metastatic disease. *Proc Natl Acad Sci USA* 89:10578-82.

Symbol ***hr***

Gene Name **hairless**

Hairless is a recessive mutation caused by retroviral integration. The gene mutated in hairless has been identified and cloned. The predicted protein product has 1182 amino acids and includes a zinc finger domain. Expression sites consonant with sites of abnormalities in hairless mutants. Mutation disrupts integrity of tissues in hair follicle.

Strain Name **HRS/J *hr/+***
Former & Common Name(s):HRS

Stock Number **000673**

Application(s) Cancer Research: Increased Tumor Incidence (Leukemia, lymphocytic) (Lymphomas, thymic) (Skin Cancers: Induced); Toxicology
Additional Research Areas
Cardiovascular Research; Dermatology Research; Immunology and Inflammation Research; Research Tools: Toxicology Research

Phenotype Mice homozygous for the *hr* spontaneous mutation have a higher incidence and earlier onset of leukemia, reducible by virus-specific antibody. Deficiency of splenic T helper cells (Ly-1+) may account for low cellular immune response of homozygous mutant mice. The coat is normal on *hr/hr* mice up to 10 days but then hair is lost from the follicle. Waves of hair growth with few thin fuzzy hairs ocur at monthly intervals for some time but homozygotes eventually become continuously hairless. Vibrissae are repeatedly regrown and shed, becoming more abnormal with age. Toenails are long and curved. There is hyperkeratosis of stratified epithelium and the upper part of hair canals beginning at 14 days. Hair club formation is abnormal. Cysts form from the hyperkeratotic upper part of hair canals and sheaths of abnormal follicles stranded in dermis. Some cysts also form from sebaceous glands. All cysts undergo sebaceous transformation and later keratinization. HRS/J mice, fed an atherogenic diet (1.25% cholesterol, 0.5% cholic acid and 15% fat), fail to develop atherosclerotic aortic lesions in contrast to several highly susceptible strains of mice (e.g. C57BL/6J, Stock No. 000664; C57L/J, Stock No. 000668, C57BR/cdJ, Stock No. 000667, and SM/J, Stock No. 000687).

Selected References
Reeve VE, Bosnic M, Boehm-Wilcox C. 1990. Effect of ultraviolet (UV) radiation and UVB-absorbing sunscreen ingredients on 7,12-dimethylbenz(a)anthracene-initiated skin tumorigenesis in hairless mice. *Photodermatol Photoimmunol Photomed* 7:222-7.

Reeve VE, Greenoak GE, Boehm-Wilcox C, Canfield PJ, Gallagher CH. 1990. Effect on topical 5-methoxypsoralen on tumorigenesis induced in albino and pigmented hairless mouse skin by UV irradition. *J Photochem Photobiol B* 5:343-57.

Symbol ***Kit^{W} Kit$^{W\text{-}v}$***

Allele Name **dominant spotting; viable dominant spotting**
Gene Name **Kit oncogene**

Strain Name **WBB6F1/J-*Kit^{W}/Kit$^{W\text{-}v}$***
Former & Common Name(s):*W ^{v};W*

Stock Number **100410**

Application(s) Cancer Research: Increased Tumor Incidence (Gonadal Tumors, ovarian); Oncogenes
Additional Research Areas
Dermatology Research; Developmental Biology Research; Endocrine Deficiency Research; Hematological Research; Immunology and Inflammation Research; Mouse Models for Human Disease; Neurobiology Research; Reproductive Biology Research; Sensorineural Research

Phenotype *Kit* mice possess pleiotropic defects in pigment-forming cells, germ cells, RBC's and mast cells. In addition, they exhibit impaired resistance to parasitic infection and an intrinsic progenitor

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cell defect. *Kit*$^{W\text{-}v}$ homozygotes resemble *Kit*^{W} homozygotes in color, anemia, and germ cells, but many of them survive to maturity. The lack of germ cells in mutant mice leads to the development of some ovarian tumors (mesotheliomas and granulosa cell), associated with an overproduction of pituitary gonadotropic hormone. *Kit*^{W}/*Kit*$^{W\text{-}v}$ double heterozygotes are viable but sterile because of germ cell deficiency. They are also mast cell deficient. *Kit*^{W}/*Kit*$^{W\text{-}v}$ double heterozygotes lack intermediate cells, derived from melanoblasts, in the stria vascularis resulting in endocochlear degeneration, loss of endocochlear potential, and hearing impairment.

Selected References

Arguello F, Furlanetto RW, Baggs RB, Graves BT, Harwell SE, Cohen HJ, Frantz CN. 1992. Incidence and distribution of experimental metastases in mutant mice with defective organ microenvironments (genotypes Sl/Sld and W/Wv). *Cancer Res* 52:2304-309.

Murphy ED. 1972. Hyperplastic and early neoplastic changes in the ovaries of mice after genic deletion of germ cells. *J Natl Cancer Inst* 48:1283-295.

Nocka K, Tan JC, Chiu E, Chu TY, Ray P, Traktman P, Besmer P. 1990. Molecular bases of dominant negative and loss of function mutations at the murine c-kit/white spotting locus: W37, Wv, W41 and W. *EMBO J* 9:1805-813.

Symbol ***Men1***

Gene Name **multiple endocrine neoplasia 1**

Strain Name **FVB;129S-*Men1*^{tm1Ctre}**

Stock Number **004066**

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Cre-lox mice require a license from DuPont.
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Application(s) Cancer Research: Increased Tumor Incidence (Adenomas, pancreatic b cells) (Cell/Tissue Type: adrenal cortical tumors) (Gonadal Tumors, ovarian and testicular) (Other Tissues/ Organs, pituitary); Tumor Suppressor Genes
Additional Research Areas
Diabetes and Obesity Research; Mouse Models for Human Disease; Research Tools: Developmental Biology Research, Endocrine Deficiency Research

Phenotype Mice that are homozygous null for the *Men1* gene die *in utero* at embryonic days 10.5-11.5, exhibiting delayed development often (20%) with defects in cranial/facial formation. At birth, heterozygous mice are viable, fertile, normal in size and do not display any gross physical or behavioral abnormalities. At nine months, ~80% of the heterozygous-null mice develop abnormalities in pancreatic islet cells, the severity of which ranges from hyperplasia to insulin-producing tumors. Parathyroid adenomas are also observed at this age. Tumor incidence is progressive, with occurrences in multiple endocrine tissues (pancreatic islets, parathyroids, thyroid, adrenal cortex, pituitary) by sixteen months of age.

Primary Reference Crabtree JS, Scacheri PC, Ward JM, Garrett-Beal L, Emmert-Buck MR, Edgemon KA, Lorang D, Libutti SK, Chandrasekharappa SC, Marx SJ, Spiegel AM, Collins F-S. 2001. A mouse model of multiple endocrine neoplasia, type 1, develops multiple endocrine tumors. *Proc Natl Acad Sci USA* 98:1118-23.

Symbol ***Prkdc*^{scid}**

Allele Name **severe combined immune deficiency**

Gene Name **protein kinase, DNA activated, catalytic polypeptide**

Mutation in the gene encoding the catalytic subunit of DNA activated protein kinase, *Prkdc*. Arose in the C.B-17 inbred strain (BALB/c.C57BL/Ka-*Igh-1*^{b}).

Strain Name **B6.CB17-*Prkdc*^{scid}/SzJ**

Former & Common Name(s):C5 7BL/6J-*Prkdc*^{scid}/SzJ; B6 scid; *scid*; *sci*

Stock Number **001913**

FEATURED MODELS

Strain Name **C3Smn.CB17-*Prkdc*^{scid}/J**

Former & Common Name(s):C3 HSmn.C-*Prkdc*^{scid}/J; C3H scid; *scid*; *sci*

Stock Number **001131**

Application(s) Research Tools: Cancer Research (B & T cell deficiency) (xenograft/transplant host)

Additional Research Areas

Immunology and Inflammation Research; Internal/Organ Research; Virology Research; Research Tools: Immunology and Inflammation Research, Toxicology Research

Phenotype Mice homozygous for the severe combined immune deficiency spontaneous mutation (*Prkdc*^{scid}, commonly referred to as *scid*) are characterized by an absence of functional T cells and B cells, lymphopenia, hypogammaglobulinemia, and a normal hematopoietic microenvironment. Normal antigen-presenting cell, myeloid and NK cell functions are strain dependent. *scid* mice carry a DNA repair defect and a defect in the rearrangement of genes that code for antigen-specific receptors on lymphocytes. Most homozygotes have no detectable IgM, IgG1, IgG2a, IgG2b, IgG3, or IgA. Thymus, lymph nodes, and splenic follicles are virtually devoid of lymphocytes. *scid* mice accept allogeneic and xenogeneic grafts making them an ideal model for cell transfer experiments. Some *scid* mice will spontaneously develop partial immune reactivity. *scid* mice that have serum Ig levels greater than 1 ug/ml are considered "leaky." *scid* leakiness is highly strain dependent, increases with age, and is higher in mice housed under non SPF conditions. In general, *scid* leakiness is high on the C57BL/6J and BALB/cBy genetic backgrounds, low on the C3H/HeJ background, and even lower on the NOD/LtSz background.

Strain Name **CBySmn.CB17-*Prkdc*^{scid}/J**

Former & Common Name(s):BA LB/cByJSmn-*Prkdc*^{scid}/J; BALB scid; *scid*;*sci*

Stock Number **001803**

Application(s) Research Tools: Cancer Research (B & T cell deficiency) (xenograft/transplant host)

Additional Research Areas

Immunology and Inflammation Research; Internal/Organ Research; Virology Research; Research Tools: Immunology and Inflammation Research, Toxicology Research

Strain Name **NOD.CB17-*Prkdc*^{scid}/J**

Former & Common Name(s): NOD/LtSz-*Prkd*^{csci}d/J; NOD scid; *scid*; *sci*

Stock Number **001303**

Application(s) Cancer Research: Increased Tumor Incidence (Lymphomas, thymic); Research Tools: Cancer Research (B & T cell deficiency) (xenograft/transplant host)

Additional Research Areas

Diabetes and Obesity Research; Immunology and Inflammation Research; Internal/Organ Research; Virology Research; Research Tools: Immunology and Inflammation Research, Toxicology Research

Phenotype Mice homozygous for the severe combined immune deficiency spontaneous mutation (*Prkdc*^{scid}, commonly referred to as *scid*) are characterized by an absence of functional T cells and B cells, lymphopenia, hypogammaglobulinemia, and a normal hematopoietic microenvironment. Normal antigen-presenting cell, myeloid and NK cell functions are strain dependent. *scid* mice carry a DNA repair defect and a defect in the rearrangement of genes that code for antigen-specific receptors on lymphocytes. Most homozygotes have no detectable IgM, IgG1, IgG2a, IgG2b, IgG3, or IgA. Thymus, lymph nodes, and splenic follicles are virtually devoid of lymphocytes. *scid* mice accept allogeneic and xenogeneic grafts making them an ideal model for cell transfer experiments. Some *scid* mice will spontaneously develop partial immune reactivity. *scid* mice that have serum Ig levels greater than 1 ug/ml are considered "leaky." *scid* leakiness is highly strain dependent, increases with age, and is higher in mice housed under non SPF conditions. In general, *scid* leakiness is high on the C57BL/6J and BALB/cBy genetic backgrounds, low on C3H/HeJ background, and even lower on the NOD/LtSz background. *Note*: BALB/cBy mice are *Igh-1*^{a} while the original C.B-17 mice are *Igh-1*^{b}.

Selected References

Beamer WG, Shultz KL, Tennent BJ, Shultz LD. 1993. Granulosa cell tumorigenesis in genetically hypogonadal-immunodeficient mice grafted with ovaries from tumor-susceptible donors. *Cancer Res* 53:3741-46.

FEATURED MODELS

Blunt T, Finnie NJ, Taccioli GE, Smith GC, Demengeot J, Gottlieb TM, Mizuta R, Varghese AJ, Alt FW, Jeggo PA, Jackson SP. 1995. Defective DNA-dependent protein kinase activity is linked to V(D)J recombination and DNA repair defects associated with the murine scid mutation. *Cell* 80:813-23.

Bosma M, Schuler W, Bosma G. 1988. The scid mouse mutant. *Curr Top Microbiol Immunol* 137:197-202.

Custer RP, Bosma GC, Bosma MJ. 1985. Severe combined immunodeficiency (SCID) in the mouse. Pathology, reconstitution, neoplasms. *Am J Pathol* 120:464-77.

Prochazka M, Gaskins HR, Shultz LD, Leiter EH. 1992. The nonobese diabetic scid mouse: model for spontaneous thymomagenesis associated with immunodeficiency. *Proc Natl Acad Sci USA* 89:3290-4.

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Christianson SW, Greiner DL, Schweitzer IB, Gott B, Beamer GL, Schweitzer PA, Hesselton RM, Shultz LD. 1996. Role of natural killer cells on engraftment of human lymphoid cells and on metastasis of human T-lymphoblastoid leukemia cells in C57BL/6J-scid mice and in C57BL/6J-scid bg mice. *Cell Immunol* 171:186-99.

Symbol ***Rag1*** *Gene Name* **recombination activating gene-1**

Strain Name **B6.129S7-*Rag1*^{tm1Mom}**

Former & Common Name(s): C5 7BL/6J-*Rag1*^{tm1Mom}; B & T cell deficient; RAG-1

Stock Number **002216**

Application(s) Cancer Research: Toxicology (B & T cell deficiency) (xenograft/transplant host); Research Tools: Cancer Research (B & T cell deficiency) (xenograft/transplant host)

Additional Research Areas

Hematological Research; Immunology and Inflammation Research; Internal/Organ Research; Research Tools: Immunology and Inflammation Research, Toxicology Research

Phenotype Mice homozygous for the *Rag1*^{tm1Mom} mutation produce no mature T cells or B cells. Their phenotype can be described as a "non-leaky" severe combined immune deficiency (*Prkdc*^{scid}/*Prkdc*^{scid}) (*Prkdc*^{scid} mice produce some B cells and IgM). They have no CD3$^+$ or T cell receptor (TCR) alpha-beta positive cells. The thymus of the mutant mice contains 15 to 130 times fewer cells than heterozygous or wildtype siblings. The thymocytes are CD8$^-$CD4$^-$ and most are IL2 receptor positive. Neither the spleen nor bone marrow contain any IgM or IgD staining cells, indicating an absence of mature B cells. These and other data suggest that B cell and T cell development has been arrested at an early stage. Macroscopically, the mutants are indistinguishable from heterozygotes or normal wildtype siblings.

Primary Reference Mombaerts P, Iacomini J, Johnson RS, Herrup K, Tonegawa S, Papaioannou VE. 1992. RAG-1 deficient mice have no mature B and T lymphocytes. *Cell* 68:869-77.

Symbol ***Terc*** *Gene Name* **telomerase RNA component**

Strain Name **B6.Cg-*Terc*^{tm1Rdp}**

Stock Number **004132**

Application(s) Cancer Research: Genes Regulating Growth and Proliferation, Increased Tumor Incidence, Tumor Suppressor Genes

Additional Research Areas

Cell Biology Research; Research Tools: Cell Biology Research, Genetics Research

Phenotype Early generation mice that are homozygous null for the *Terc* gene are phenotypically normal. No *Terc* transcript or telomerase activity is detected. If null mice are maintained as homozygotes, progressive adverse effects on the reproductive and hematopoietic systems are observed. By the fifth generation of homozygous intercrossing, fertility is significantly diminished. Testes size and weight is reduced by ~80%. Germ cells exhibit decreased rates in proliferation and increased rates of apoptosis resulting in a general state of germ cell depletion. Females exhibit smaller ovaries and diminished uterine horns. The proliferative

capacity of hematopoietic cells derived from bone marrow and spleen is significantly compromised. Cell cultures of primary embryonic fibroblasts derived from null embryos exhibit telomere shortening (4.8 +/- 2.4 kb per generation). Cells from the fourth generation onward possess chromosome ends lacking detectable telomere repeats, aneuploidy, and chromosomal abnormalities, including end-to-end fusions.

Primary Reference Blasco MA, Lee H-W, Hande MP, Samper E, Lansdorp PM, DePinho RA, Greider CW. 1997. Telomere shortening and tumor formation by mouse cells lacking telomerase RNA. *Cell* 91:25-34.

Symbol ***Vhlh***

Gene Name **von Hippel-Lindau syndrome homolog**

Strain Name **C;129S-*Vhlh*^{tm1Jae}**

Former & Common Name(s): *Ho xb4*

Stock Number **004081**

Licensing Cre-lox mice require a license from DuPont.

Application(s) Cancer Research: Tumor Suppressor Genes

Phenotype This strain contains *loxP* sites flanking the *Vhlh* promoter and exon 1 resulting in a conditional null allele. Mice that are homozygous for this allele are viable, fertile, normal in size and do not display any gross physical or behavioral abnormalities. Cre-mediated recombination results in the deletion of the promoter and exon 1. Studies in which liver-specific inactivation of the *Vhlh* gene was achieved by breeding this strain with albumin promoter driven-Cre mice resulted in hemizygous mice that exhibit cavernous hemangiomas of the liver, a rare component of the human von Hippel-Lindau (VHL) disease. This strain represents an effective tool for generating tissue specific-targeted mutants that would be useful in studies examining VHL and tumor suppression in general.

Primary Reference Haase VH, Glickman JN, Socolovsky M, Jaenisch R. 2001. Vascular tumors in livers with targeted inactivation of the von Hippel-Lindau tumor suppressor. *Proc Natl Acad Sci USA* 98:1583-8.